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Inactivation of Feline Calicivirus and Adenovirus Type 40 by UV Radiation

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Little information regarding the effectiveness of UV radiation on the inactivation of caliciviruses and enteric adenoviruses is available. Analysis of human calicivirus resistance to disinfectants is hampered by the lack of animal or cell culture methods that can determine the viruses' infectivity. The inactivation kinetics of enteric adenovirus type 40 (AD40), coliphage MS-2, and feline calicivirus (FCV), closely related to the human caliciviruses based on nucleic acid organization and capsid architecture, were determined after exposure to low-pressure UV radiation in buffered demand-free (BDF) water at room temperature. In addition, UV disinfection experiments were also carried out in treated groundwater with FCV and AD40. AD40 was more resistant than either FCV or coliphage MS-2 in both BDF water and groundwater. The doses of UV required to achieve 99% inactivation of AD40, coliphage MS-2, and FCV in BDF water were 109, 55, and 16 mJ/cm^2, respectively. The doses of UV required to achieve 99% inactivation of AD40, coliphage MS-2, and FCV in groundwater were slightly lower than those in BDF water. FCV was inactivated by 99% by 13 mJ/cm^2 in treated groundwater. A dose of 103 mJ/cm^2 was required for 99% inactivation of AD40 in treated groundwater. The results of this study indicate that if FCV is an adequate surrogate for human caliciviruses, then their inactivation by UV radiation is similar to those of other single-stranded RNA enteric viruses, such as poliovirus. In addition, AD40 appears to be more resistant to UV disinfection than previously reported.

Disinfection is an important treatment barrier between drinking water consumers and viral gastroenteritis, since the small size (~25 to 80 nm) of enteric viruses may permit their passage through conventional filtration processes (9). Concern over the formation of chlorinated organics and the effectiveness of chemical disinfectants has increased interest in the drinking water and wastewater industries to consider alternative disinfectants. One option that is receiving increased attention and produces few disinfectant by-products is UV radiation (12).

Members of the human *Calicivirus* genus, "Norwalk-like viruses" (NLVs), are an important cause of acute gastroenteritis outbreaks in adults and children throughout the world (10). The etiological agents of over half of all reported outbreaks of disease go unrecognized, but the outbreaks are thought to be caused by viral agents. Survival in ice and at 60°C has demonstrated the NLVs' increased environmental stability (10). These viruses are now considered to be the second most common cause of viral gastroenteritis in children, following rotavirus (10). Like NLVs, the enteric adenoviruses 40 (AD40) and 41 (AD41) are also important causes of self-limiting, acute gastroenteritis in children (<4 years of age) (17, 20). NLVs and enteric adenoviruses are shed in feces, low numbers of particles can produce illness, and prolonged shedding is typical (10, 17). Although no waterborne outbreaks have been reported for the enteric adenoviruses, increased environmental stability compared to those of other enteric viruses and their presence in sewage and surface waters have been reported (8, 25, 29). Outbreaks from waterborne viruses contracted by swimming have been reported for the non-enteric adenoviruses (7, 22).

Both caliciviruses and enteric adenoviruses are on the U.S. Environmental Protection Agency's Drinking Water Contaminant Candidate List (CCL) (30). These viruses are on the CCL for regulatory consideration, since little to no information regarding health, analytical methodologies, or drinking water and wastewater treatment is currently available. Very few studies have been conducted on the inactivation of these viruses by water disinfectants such as UV light. The objectives of this study were to determine the inactivation kinetics of AD40 and feline calicivirus (FCV) in two water types—buffered-demand-free (BDF) water and treated drinking waters (groundwater and surface water)—after exposure to low-pressure UV radiation. Since there are currently no known animal or mammalian cell culture systems that can determine infectivity of NLVs, a closely related surrogate, FCV, was used in the present study as a model for NLV inactivation by UV light. FCV has been suggested to be an adequate surrogate for NLV inactivation, since it has a similar genome organization and capsid architecture (5, 28). The inactivation kinetics of coliphage MS-2, a suggested indicator of viral inactivation by UV light, were also determined and compared to those of FCV and AD40 (15).

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MATERIALS AND METHODS

Virus propagation and assay. AD40 (strain Dugan), FCV (strain F9), the primary liver carcinoma cell line PLC/PRF/5, and the Crandell feline kidney (CRFK) cell line were obtained from the American Type Culture Collection (Manassas, Va.). AD40 and FCV were propagated and assayed in the PLC/PRF/5 and CRFK cell lines (5, 6, 11). Maintenance medium (minimum essential medium containing 10% fetal bovine serum) was decanted from 162-cm^2 tissue culture flasks containing complete monolayers. The flasks were rinsed with sterile Tris-buffered saline (Trizma base) (Sigma Chemical Co., St. Louis, Mo.), and 1 ml of an approximately 10^4 to 10^5 most-probable-number (MPN) concentration of the viral stock per ml was inoculated onto the monolayer. In order to allow virus attachment, the flasks were incubated for 1 h at room temperature. The flasks were rocked every 15 min throughout the 1-h incubation. After incubation, 50 ml of maintenance medium (without fetal bovine serum) was inoculated onto the infected cell monolayer, and the mixture was incubated at 37°C until at least 90% of the cell monolayer had been destroyed. The cell culture flasks containing the propagated virus were frozen and thawed: one time for AD40 and three times for FCV. Next, the supernatant was centrifuged at 10,000 × *g* at 4°C to remove cell debris.

Further purification and concentration of FCV and AD40 were accomplished by performing two successive polyethylene glycol (PEG) precipitation procedures. Briefly, for every 100-ml volume of viral supernatant, 9 g of PEG (molecular weight, 8,000) and 5.8 g of NaCl were added and stirred overnight at 4°C. Centrifugation at 10,000 × *g* at 4°C for 30 min was performed, followed by disposal of the supernatant and resuspension of the pellet in BDF water. BDF water was prepared by dissolving 0.54 g of Na_2HPO_4 (anhydrous) and 0.88 g of KH_2PO_4 (anhydrous) per liter of purified water (Nanopure RO purifier; Barnstead, Dubuque, Iowa) and was adjusted to pH 7 with 1 M NaOH and 1 M KH_2PO_4. For dispersion of FCV and AD40 stocks, equal volumes of the viral extracts and chloroform were homogenized by vortexing the mixture for at least 10 min. Once homogenized, the chloroform-virus suspension was centrifuged at 10,000 × *g* at 4°C for 15 min. After centrifugation, the upper layer containing the purified viral stock was collected and stored at 4°C.

Determination of viral titer before and after UV radiation was accomplished by plating 5- or 10-fold dilutions in quadruplet in a 24-well tissue culture tray with the appropriate cells in suspension. The concentration of the viruses was determined by observation of each infected well for cytopathic effect (CPE). Observation for CPE was continued up to 12 and 24 days for FCV and AD40, respectively. The MPN General Purpose Program was used to determine the concentrations (MPN per milliliter) of both AD40 and FCV (18).

Propagation of MS-2 coliphage was accomplished by the double-layer agar technique (1). An 18- to 24-h culture of *Escherichia coli* (ATCC 15977) was grown in tryptic soy broth (TSB; Difco, Detroit, Mich.), and 1 ml of this culture was transferred to fresh TSB and grown for 3 h at 37°C in a shaking water bath. MS-2 stock was serially diluted in BDF water in order to achieve a final concentration of 10^5 PFU/ml. A 1-ml suspension of the host cells and 0.1 ml of the phage dilution were mixed in 3 ml of molten overlay agar and poured on petri dishes containing tryptic soy agar (TSA; 1.5% agar; Difco). After 24 h of incubation at 37°C, the bacteriophage was eluted by the addition of approximately 6 ml of BDF water and incubation at room temperature for 1 to 3 h. After incubation, the liquid from the plates was collected and centrifuged at 10,000 × *g* for 10 min to remove bacterial debris. Further purification included chloroform extraction, as described for FCV and AD40 processing. Stock phage was serially diluted in BDF water in order to achieve a concentration that would allow observation of at least 99.99% inactivation for all UV disinfection experiments. Phage samples from the UV radiation experiments were serially diluted in BDF water and plated in duplicate by the double-agar technique as described previously. The resulting plaques were enumerated and averaged in order to determine MS-2 coliphage survival.

UV lamp setup. The collimated beam apparatus consisted of an 8-W low-pressure mercury vapor germicidal lamp (Sankyo Denki, G8T5.2N) that emitted nearly monochromatic UV radiation at 253.7 nm and was suspended horizontally in a wood box. For uniform lamp output, the lamp was warmed up for at least 30 min prior to all experiments. Attached to the UV lamp box was the collimating tube, made of black polyvinyl chloride pipe measuring 53.3 cm long and with a diameter of 7.6 cm. Both the UV box and collimating tube were painted with a nonreflective black paint to minimize light reflection. A stir plate was placed directly under the collimated beam for slow stirring of the viral suspensions. The UV intensity of each experiment was determined with a calibrated UV 254-nm detector (IL-2000, photodetector SED 240/NS254/W; International Light, Newburyport, Mass.) by placing the radiometer at approximately the same location and elevation as the water surface of the irradiated samples.

TABLE 1. Water quality parameters for BDF water and treated groundwater

Water type	Water source	pH	Turbidity (NTU)[a]	A_{254}
BDF	NA[b]	7.0	<0.001	<0.001
Groundwater[c]	Great Miami Aquifer	8.0	0.1–0.3	0.011

[a] NTU, nephelometric turbidity unit.
[b] NA, not applicable.
[c] Treated (dechlorinated) groundwater.

Test waters. For all viruses, at least two experiments were performed with BDF water at pH 7 and room temperature (22 to 25°C). The effects of UV radiation on viral inactivation in treated (coagulation, sedimentation, chlorination, and filtration) groundwater were also determined. The groundwater was dechlorinated by rapid mixing and exposure to UV light until no chlorine was measured by the *N*,*N*-diethyl-*p*-phenylenediamine (DPD) method (2). Two experiments were performed with FCV and AD40 suspended in the treated groundwater sample. Table 1 lists the properties of each water type.

Irradiation of samples. Samples were irradiated in sterile Pyrex (60 by 15 mm) glass petri dishes containing stir bars (10 by 2 mm) stirring at low speed. The viral stock was diluted in either BDF water or one of the two treated drinking waters in order to achieve total inactivation levels of 99 and 99.99% for AD40 and FCV, respectively. The total volume and depth of viral suspension within the petri dish were 14 ml and 1 cm, respectively. Prior to each test, the A_{254} of the viral suspension was measured (Spectronic Genesis 5 spectrophotometer; Milton Roy Co., Rochester, N.Y.). Viral suspensions were placed under the collimated beam and irradiated for predetermined times. Samples of 1 to 4 ml were withdrawn for viral assay. In addition, control samples, not subjected to UV light, were collected at the same time. Both the initial and final sampling times were withdrawn from the control petri dishes in order to determine the concentration of viruses in the viral suspension and to determine if viral inactivation occurred in the absence of UV radiation. Temperature and pH were also determined for each experiment. Each experiment was conducted at least in duplicate, with the exception of the FCV inactivation experiment carried out in surface water.

Dose determination. According to Beer's law, the measured intensity was corrected for absorbance of the liquid sample with the following equation:

$$I_{ave} = I_o(1 - e^{-aeL})/a_eL$$

where I_{ave} = intensity average (milliwatts per square centimeter), a_e = absorbance of the suspension to the base e, L = depth (centimeters) of the solution irradiated by the collimating beam, and I_o = average of the measured intensity at time zero (t_0) and at the final time (t_{final}). The UV dose was calculated as the product of the average intensity (milliwatts per square centimeter) multiplied by the time (seconds) of UV exposure in order to achieve 90, 99, 99.9, or 99.99% viral inactivation. UV dose units can be expressed as milliwatts per square centimeter or millijoules per square centimeter, since the two are equal.

Viral inactivation analysis. Since the inactivation of viruses has been shown to be of a first-order type, Chick's law can be used to describe the kinetics of viral inactivation by UV (27). Viral UV inactivation can be defined by the following equation: $N_t/N_0 = e^{-kit}$, where N_t = the number of viral particles at time t (time of UV exposure), N_0 = the number of viral particles at time zero (no UV radiation applied), k = inactivation rate or slope of inactivation curve, i = intensity of UV light energy (milliwatts per square centimeter), and t = exposure time (seconds). The parameter $-\log_{10}$ of the survival ratio (N_t/N_0) versus dose for each experiment was used to perform regression analysis for each virus and water type by using Quatro Pro (version 9, 1999; Corel Corporation). Generation of regression curves and prediction of the doses required for 90, 99, 99.9, or 99.99% viral inactivation were accomplished by including data points from all experiments for each virus and water type. The predicted (linear line) and observed (individual data points) values were plotted on charts (Quatro Pro, version 9, 1999), representing log inactivation [$-\log(N_t/N_0)$] versus dose, for each set of UV disinfection experiments involving one of the tested viruses suspended in either BDF or treated drinking water (Fig. 1 and 2).

RESULTS

UV light inactivation curves for FCV, MS-2, and AD40 for all replicate experiments carried out in BDF water and treated

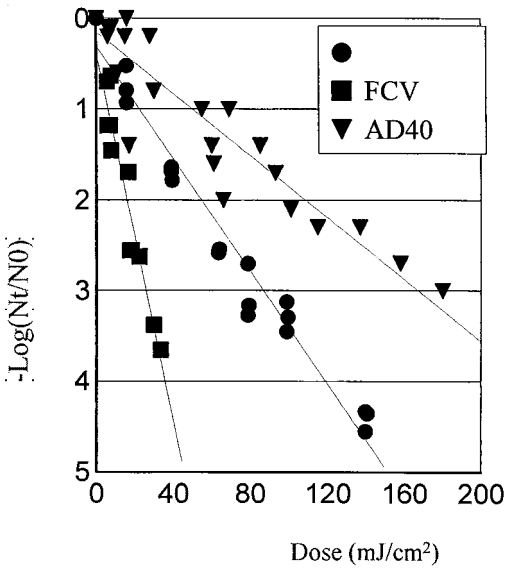

FIG. 1. UV light inactivation of AD40, FCV, and MS-2 in BDF water.

groundwater are shown in Fig. 1 to 3. Table 1 includes the turbidity, pH, and UV light absorbance at 254 nm for each water type. The doses for 90 to 99.99% inactivation for AD40, FCV, and MS-2 are listed in Table 2, and the associated R^2 values and inactivation constants are listed in Table 3. All calculations in these tables were based on all replicate experiments for each virus. Unlike FCV and MS-2, 99.99% inactivation was not achieved in any of the AD40 inactivation experiments due to difficulties in propagating high-titer viral stocks. Instead, AD40 UV light inactivation ranged from 1 to 3 logs.

FCV and MS-2 exhibited typical first-order inactivation kinetics. Tailing or flattening was not observed for any of these inactivation curves, indicating a lack of viral clumping or aggregation of the MS-2 and FCV prepared stock suspensions. Alternatively, the majority of the AD40 inactivation curves demonstrated slight shouldering or flattening for the UV inactivation curves at UV doses of <50 mJ/cm^2, followed by linear reduction (Fig. 3). Compared to what occurred in the first three experiments performed under the same conditions, the fourth AD40 experiment, conducted in BDF water, displayed slower inactivation kinetics and slight tailing seemed to occur. If experiment 4 was considered an outlier and therefore deleted from the data set, regression analysis of experiments 1 to 3 would have higher R^2 values than those of calculations that included all four experiments. The doses required for 90, 99, 99.9, and 99.99% inactivation, calculated on the basis of the regression analysis of experiments 1 to 3, are 57, 113, 170, and 226 mJ/cm^2, respectively.

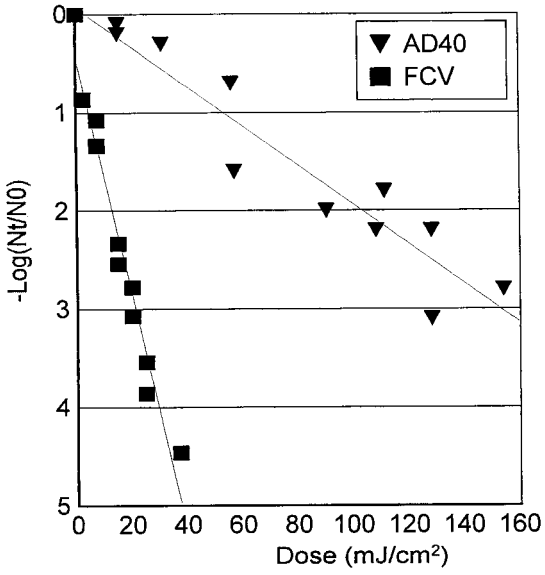

FIG. 2. UV light inactivation of FCV and AD40 in treated drinking water.

TABLE 2. UV doses for 90 to 99.99% inactivation of test viruses in BDF water and groundwater at room temperature

Virus	Water type[a]	UV dose (mJ/cm^2) for inactivation level			
		90%	99%	99.9%	99.99%
MS-2	BDF	23	55	87	119
FCV	BDF	6	16	26	36
	Ground	5	13	21	29
AD40	BDF	50	109	167	226[b]
	Ground	53	103	153	203[b]

[a] Ground, treated (dechlorinated) groundwater.
[b] Extrapolated value, not achieved in bench-scale experiments, based on linear regression.

AD40 was the virus most resistant to UV light inactivation in both water types tested. In BDF water, AD40 was the most resistant virus, followed by MS-2 and FCV. In groundwater, FCV was more susceptible to inactivation than AD40. Similar UV light doses were observed for inactivation experiments carried out in treated groundwater compared to BDF water. A dose of 6 mJ/cm^2 was needed to inactivate FCV in BDF water by 90%, whereas 5 mJ/cm^2 was required for the same amount of inactivation in treated groundwater. The treated groundwater had a higher UV absorbance than the BDF water (Table 1), yet little difference was observed between the doses needed to achieve 90 to 99.99% AD40 and FCV inactivation in these two water types (Table 2). MS-2 disinfection experiments were not conducted with treated groundwater.

TABLE 3. Inactivation constant, standard error, and R^2 values obtained by regression analysis for UV disinfection experiments

Virus	Total no. of expts	K^a	SE of K	R^2	Water type[b]
MS-2	3	0.0310	0.0013	0.9680	BDF
FCV	3	0.1016	0.0101	0.9187	BDF
FCV	2	0.1217	0.0079	0.9597	Ground
AD40	4	0.0171	0.0013	0.8820	BDF
AD40	2	0.0201	0.0012	0.9217	Ground

[a] K, inactivation constant.
[b] Ground, treated (dechlorinated) groundwater.

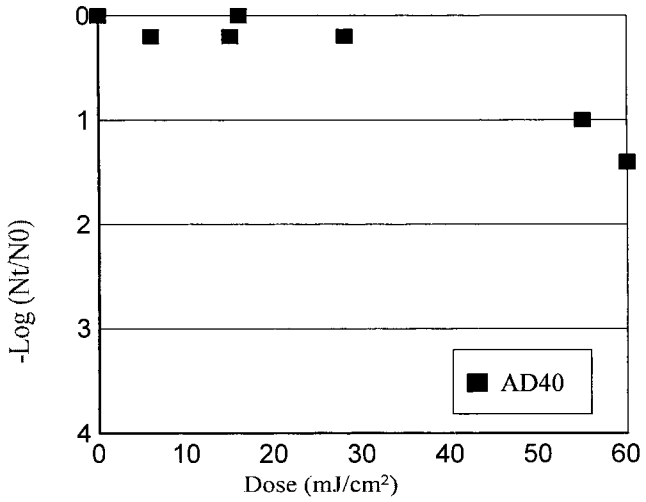

FIG. 3. Shouldering effect of AD40 at low doses in BDF water.

DISCUSSION

The UV doses commonly applied for water and wastewater treatment are between 30 and 40 mJ/cm^2, and the National Sanitary Foundation (NSF) has increased UV water treatment standards for class A point-of-entry and point-of-use to 40 mJ/cm^2 (ANSI/NSF standard 55 [2a]). Under these standards, FCV would be adequately reduced ≥99.99% in water supplies. Higher doses would be required to reduce AD40, since 40 mJ/cm^2 would not be adequate for even 90% reduction.

Currently no laboratory methods are available for the growth and propagation of NLV in the laboratory, but the closely related FCV has been used as a model system in previous inactivation studies involving physical and chemical disinfectants (5, 28). The results of this study indicate that UV inactivation of FCV is similar to that of other single-stranded RNA (ssRNA) viruses, such as poliovirus type 1 (PV-1). The doses required for 90, 99.9, and 99.99% inactivation of FCV in BDF water were 6, 26, and 36 mJ/cm^2, respectively. Wolfe (32) and Wilson et al. (B. R. Wilson, P. F. Rossler, E. Van Dellem, M. Abbaszadegan, and C. P. Gerba, Poster, Proc. Am. Water Works Assoc.-Water Qual. Technol. Conf., Toronto, Canada, 1992, p. 219–235) reported doses of 5 and 7.7 mJ/cm^2 for 90% inactivation of PV-1, respectively, whereas Hill et al. (16) determined that a dose of 30 mJ/cm^2 was necessary for 99.9% inactivation. The results of this study indicate that properly operated low-pressure UV disinfection systems should be capable of eliminating the threat of this pathogen in water.

Respiratory and enteric adenoviruses are considerably more resistant to UV radiation than other double-stranded DNA (dsDNA) and ssRNA viruses. Hara et al. (13) determined that AD19 is 60 times more resistant than EV70 (ssRNA) to low-pressure UV radiation. Cameron et al. (4) reported increased UV resistance of adenovirus (AD5) compared to other dsDNA viruses (simian virus 40 and herpes simplex virus type 1). In addition, Wasserman (31) reported that AD4 and -20 were more resistant to UV light than AD1, -19, and -24, demonstrating varied susceptibilities between adenovirus types. This was also demonstrated by the research carried out by Cameron (4), where AD5 was more resistant than AD7 to UV light inactivation (4). In the present study, doses required to achieve 90 to 99.99% inactivation of AD40 were higher than those previously reported for other enteric viruses and adenoviruses.

The exceptionally greater UV resistance of AD40 than FCV and other viruses may be due to nucleic acid composition. Adenoviruses have dsDNA as their genetic material. It has been suggested that viruses with double-stranded genomes are less susceptible to UV inactivation, since only one strand of the nucleic acid may be damaged during disinfection. The undamaged strand may then serve as a template for repair by host enzymes (14, 19). For DNA viruses, host cells can contain the enzymatic machinery to repair damage by excision or recombinational repair. This has been suggested as a reason for the shouldering effect observed in UV disinfection experiments concerning dsDNA viruses (14). Host cell mechanisms for the repair of viral RNA genomes may not be as likely, since excision has not been demonstrated, and may explain the increased susceptibility of FCV over AD40 (14). Multiple infection of host cells, also termed "multiplicity of reactivation," can lead to enhanced survival of either DNA or RNA viruses (3, 14, 27). Multiplicity of reactivation has been proposed as the mechanism for enhanced survival after UV disinfection of several viruses, including poliovirus and adenovirus (14, 27).

Small proteins concentrated along with viral particles in prepared viral stocks or characteristics of the architecture of viral capsids can influence the effectiveness of UV radiation and may be characterized by shouldering, flattening, or tailing of inactivation curves. For adenovirus, only 10 to 20% of the viral structural polypeptides are assembled into new adenovirus particles, which may explain the low titer produced by this virus (23). These viral precursors, which would be present in the prepared virus stock, may consume or shield UV radiation from infectious adenovirus particles. The formation of defective viral particles has not been demonstrated for caliciviruses. Shielding or consumption of UV radiation before reaching the nucleic acid may also occur because of the presence of capsid proteins or other packaged viral proteins that are directly associated with the nucleic acid. The adenovirus capsid is complex, consisting of several capsid proteins and protruding protein fibers, whereas the calicivirus capsid lacks fibers and is made up of only one viral protein (23, 24). The more complex adenovirus capsid may afford additional protection from UV radiation over a simple capsid architecture, like that found with caliciviruses. Due to the AD40 capsid's complex structure, multiple hits of UV radiation may be required for irreparable damage of AD40 viral particles.

Since the mechanisms of AD40 UV light inactivation are complicated (UV light protection from polypeptides in viral preparation, complex nucleic acid and capsid structures, and host cell repair mechanisms), varied inactivation kinetics between replicate experiments are likely. Therefore, AD40 inactivation calculations were based on all replicate experiments performed. The R^2 values of each set of data ranged from 0.88 to 0.97 for AD40, FCV, and MS-2, where the lowest values were observed for AD40 experiments conducted in BDF water. Small differences between viral preparations may be the cause of the variations in UV doses required for viral inactivation between replicate experiments. Viral preparations that are more pure (i.e., containing very little cellular debris or viral

noninfectious peptides) are more likely to require lower UV doses for viral inactivation due to increased penetration of UV light. Furthermore, variations in the efficiency of host cell repair, in the case of AD40, of the viral genome may also be the cause of the differences in UV light inactivation among experimental replicates. Of the four AD40 experiments performed with BDF water, experiment 4 had considerably lower inactivation kinetics than the first three experiments. Due to the apparent deviation of the inactivation kinetics of experiment 4, an additional regression analysis calculation excluding experiment 4 was performed. As expected, regression analysis of experiments 1 to 3 produced higher R^2 values than those found with calculations that included all four experiments. The doses required for 90, 99, 99.9, and 99.99% inactivation, calculated on the basis of experiments 1 to 3, are 57, 113, 170, and 226 mJ/cm^2, respectively. Even when considering only experiment 4, only 2 logs of inactivation occurred at a UV light dose of 66 mJ/cm^2. The inactivation kinetics of this single experiment are still less than those observed for MS-2 and FCV. No matter how the data are interpreted, all results demonstrate that AD40 has increased resistance to UV light inactivation.

The doses needed to inactivate AD40 were higher in the present study than those in previous work conducted by Meng and Gerba (21). Meng and Gerba reported 30 and 124 mJ/cm^2 required for 90 and 99.99% inactivation of AD40, respectively, whereas higher doses of 50 (measured value) and 203 (extrapolated value) mJ/cm^2 were observed in this study. Differences in viral preparation methodologies between the two studies may be one explanation for the variation in AD40 UV light inactivation kinetics. Previous work in our laboratory has demonstrated that the enteric adenoviruses are sensitive to successive freeze-thawing. The protocols carried out by Meng and Gerba (21) included five freeze-thaw steps, which may have increased viral susceptibility to UV light inactivation by, for example, weakening the viral capsid. Only one freeze-thaw step was performed in the present study. Like those reported between this study and that of Meng and Gerba (21), variations in the kinetics of inactivation between similar inactivation studies must be carefully examined, especially if the results will be applied for regulatory purposes. The methods used in microbial preparation, water type, and experimental design can produce significant differences in inactivation kinetics or do not reflect microbial inactivation during water treatment. Nonetheless, both studies demonstrate that enteric adenoviruses are extremely resistant to UV disinfection compared to other enteric viruses.

For both FCV and AD40, the doses required for 90 to 99.99% inactivation were similar for both BDF water and groundwater experiments. The higher turbidity measurements of the treated groundwater did not increase the resistance of the tested viruses to UV radiation; instead, the doses were similar to those observed for BDF water. This is surprising, since treated groundwater components may decrease UV light penetration or shield viruses from irradiation. Instead, constituents within the treated groundwater may have enhanced the effects of UV light, thereby increasing viral inactivation rates.

In the present study, 90 and 99.9% inactivation of coliphage MS-2 required doses of 23 and 87 mJ/cm^2, respectively. Similarly, Battegelli et al. (3) reported a dose requirement of >25 mJ/cm^2 for 90% inactivation of coliphage MS-2, although Wilson et al. (Poster, Water Qual. Technol. Conf., Toronto, Canada, 1992) and Meng and Gerba (21) reported lower dose requirements of 18.6 and 14 mJ/cm^2, respectively. The doses observed in this study for coliphage MS-2 were over 3 times higher than those for FCV and over 2 times lower than those observed for AD40 inactivation in BDF water. Although coliphage MS-2 has been suggested as an adequate indicator for enteric virus UV inactivation, these results suggest that it would not be an acceptable indicator for UV inactivation of AD40. However, coliphage MS-2 may serve as an adequate, conservative indicator for the UV inactivation of FCV.

The results of this study provide information on the effectiveness of UV radiation of emerging viral enteric pathogens (with FCV as a surrogate for human caliciviruses) in waters with different physical qualities. Susceptibility of the virus to UV light is highest for FCV, followed by MS-2 and AD40 in absorbance-free (BDF) water and in treated groundwater containing constituents that absorb UV light. In groundwater, the kinetics of AD40 and FCV inactivation were not decreased by this water's constituents. According to this study, at least 99.99% inactivation would occur for FCV at the NSF-recommended dose of 40 mJ/cm^2; however, not even 90% inactivation of AD40 would be achieved. This study's results provide a basis for the establishment of drinking water treatment guidelines for proficient application of low-pressure UV radiation for inactivation of calicivirus and adenovirus in low-UV-light-absorbing waters.

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